

**МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
ВИЩИЙ ДЕРЖАВНИЙ НАВЧАЛЬНИЙ ЗАКЛАД УКРАЇНИ
«БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»**



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101 – ї

підсумкової наукової конференції

професорсько-викладацького персоналу

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secreted in the dark. Even a slight lighting inhibits its synthesis. Melatonin has been shown to have a wide range of biological effects, but its main feature is a powerful antioxidant action.

The objective of the work was to study the level of ceruloplasmin in the rats' blood plasma under subacute alcohol intoxication, its combination with light exposure and melatonin administration.

The experiments were conducted on 32 albino male rats with body weight of 180-230 g. A subacute alcohol intoxication was induced by intragastric administration of 40% ethanol in the dose of 7 ml/kg of the body weight for 7 days. A light exposure was caused by keeping animals under a fluorescent light of 1500 lux intensity for 24 hours a day.

Alcohol intoxication along with the permanent light exposure were found to cause a significant increase in the ceruloplasmin concentration in blood plasma by 88% above the control. This parameter was higher than that of rats which had alcohol intoxication induced under normal light regime by 80% and which might have been resulted from decrease in melatonin synthesis and lack of its antioxidant effect under constant light exposure.

The administration of melatonin at the dose of 5 mg / kg daily at 20⁰ for 7 days to animals exposed to ethanol intoxication or its combination with constant lighting prevented elevation of ceruloplasmin level in blood plasma. Animals that were administered melatonin against the background of the combination of alcohol intoxication with light exposure showed a tendency to normalization of ceruloplasmin level, but the figure remained 24% above the control.

Thus, the administration of melatonin against the ground of alcohol intoxication or its combination with constant light exposure contributed to normalization of blood plasma ceruloplasmin which proves melatonin's antioxidant properties.

Dikal M.V.

BIOCHEMICAL CHANGES OF BLOOD PLASMA INDICATORS IN THE MODELING OF ALOXAN DIABETES IN RATES

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The number of patients with diabetes mellitus (DM) in the world is growing steadily. At present there is no clear molecular mechanisms of inheritance of type I diabetes mellitus, and there is no single conventional theory that would explain the numerous data obtained in this field. And various metabolic lesions of systems and organs that develop against the background of type II diabetes are a significant threat to health and one of the causes of disability of the population.

The experiment was conducted on 40 non-linear male rats weighing 0.16-0.18 kg, which were divided into two subgroups: control intact rats (n = 20) and experimental (n = 20) rats with induced alloxan DM, which was caused by the introduction of 5% alloxan monohydrate intraperitoneally in the dose of 150 mg/kg. Basal glycemia studies were performed using a One Touch device (manufactured by Johnson & Johnson), which was ≥ 10.0 mmol/l for blood sampling from the tail vein. All manipulations with animals were carried out according to European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes and law of Ukraine "On protection of animals from cruelty". The material for the study was blood plasma. The concentration of the main biochemical parameters was determined in heparinized plasma without traces of hemolysis by conventional methods.

Against the ground of significant hyperglycemia (12.6 ± 1.28 mmol / l) the concentration of total protein of blood of experimental animals was found to decrease significantly ($p < 0.05$) and against the ground of disturbance of transamination and metabolism of amino acids, in the absence of significant change in albumin concentration. Such changes may be indicative of certain imbalance in the synthesis of the globulin fraction and inform redistribution of the content of many acute-phases proteins. However, urea and creatinine concentrations remained unchanged when comparing the control and experimental groups on the 14th day of the disease simulation.



Thus, alloxan toxic effect was proved to occur not only concerning the pancreas, but also leads to change of the main biochemical processes in hepatocytes, namely, the hepatic protein-synthetic function, which indicates a decrease in the total protein content (51.1 ± 0.85 g/l) due to the generation of a large number of toxic metabolites, including free radicals, which resulting in development of oxidative stress.

Gerush I.V.

GLUTATHIONE EFFECT ON HYDROGEN SULFIDE LEVEL IN THE BLOOD BY EXPERIMENTAL NEPHROPATHY

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The incidence of renal disease has increased steadily in recent years. Structural modifications in the kidney may cause oxidative stress related to an imbalance between free radical production and antioxidant capacity. Free radicals formed with oxidative stress, destroy lipids and proteins on the membranes and cause modifications and oxidation of lipids and proteins thereby damaging cells. Currently, therapeutic options available for managing renal diseases are not quite efficient and therefore, there lies a subsequent need for effective therapies that can prevent progressive damage to the kidneys.

Recent researches show that hydrogen sulfide (H₂S) via antihypertensive, antioxidative, antiapoptotic and anti-inflammatory mechanisms takes part in the protection of the kidney. And our study was designed to investigate the effect of glutathione on H₂S level in the blood of rats with experimental nephropathy.

The experiment was conducted on 131 male albino rats with the body weight 0.16 - 0.18 kg. Experimental nephropathy was modeled by injection of a single intraperitoneal dose of folic acid (250 mg/kg, (Sigma-Aldrich)). In order to confirm pathology the kidneys were examined by means of morphometric analysis. Glutathione (Sigma-Aldrich) was introduced daily (100 mg/kg) by intragastric way for 3 and 7 days following after the injection of folic acid. Animals were divided into 5 groups: I – control group (n=36), II – nephropathy (3^d day (n=25)), III – nephropathy + 3 days of glutathione introduction (n=23), IV – nephropathy (7th day (n=24)), V – nephropathy + 7 days of glutathione introduction (n=23). Rats were kept under the standard vivarium conditions at constant temperature and basic allowance. Animals were narcotized with chloroform and sacrificed on the next day after the last glutathione introduction. All manipulations with animals were carried out according to European Convention for the Protection of Vertebrate Animals used for Experimental and Other Scientific Purposes and law of Ukraine “On protection of animals from cruelty”. Plasma concentration of H₂S was measured using spectrophotometer Agilent Cary 60. The type of distribution was estimated using Shapiro-Wilk test. Significant differences between group were evaluated by using Wilcoxon test with $p < 0.05$ considered. All the results in figures are represented as median minimum-maximum values (Me[*min-max*]).

H₂S levels in the blood of rats with nephropathy were lower by 35.5% on the 3d day and by 25.7% on 7 days of the experiment than those in control rats. Blood H₂S level on 3 experimental day was associated with a specific volume of epithelial cells of proximal tubules of the kidney. In particular, the specific volume of epitheliocytes of proximal tubules in the state of alteration is 84.8%. Oxidation modification of proteins increases (according to the R/B coefficient stained with bromophenol blue for Mikel Calvo). Glutathione increases the level of gasotransmitter by 14.3 % in blood plasma of rats on 3 day and by 11 % on 7 day of the experimental period compared with the group of animals without treatment.

Taken together, our studies show the existence of dependence between the concentration of hydrogen sulfide in the blood and development of kidney disease and confirm protective and antioxidant properties of glutathione.